

Case Report

Ablation of Idiopathic Ventricular Tachycardia with a Left Bundle-Branch Block Morphology Originating from the Pulmonary Artery

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We successfully performed radiofrequency catheter ablation (RFCA) in 2 cases involving patients with idiopathic ventricular tachycardias (VTs) and premature ventricular contractions (PVCs) originating from the pulmonary artery (PA). The QRS morphology of the VTs and PVCs in the two cases exhibited a left bundle-branch block (LBBB) morphology with an inferior axis. Activation and pace mappings were performed in the right ventricular outflow tract (RVOT) and above the pulmonary valve to determine the origin of the VTs and PVCs. In both cases, the earliest ventricular activation was recorded in the PA above the pulmonary valve. Applications of radiofrequency current at those sites in the PA resulted in the elimination and noninducibility of the VT and PVC. During the follow-up, the VT or PVC did not recur without any antiarrhythmic drug administration.

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Key words: Pulmonary valve, Premature ventricular contractions, Activation mapping, Pace mapping, CARTO

Introduction

Radiofrequency catheter ablation (RFCA) is an established curative therapy for idiopathic ventricular tachycardias (VTs) or symptomatic premature ventricular contractions (PVCs) with a left bundle-branch block (LBBB) morphology originating from the outflow tract (OT) in structurally normal hearts. Most of those arrhythmias have their origin located

on the septal side of the right ventricular outflow tract (RVOT). However, some originate from the free wall of the RVOT, left ventricular outflow tract (LVOT) or aortic sinus cusp (ASC). Additionally, several reports have indicated that some idiopathic VTs originate from the pulmonary artery (PA) and can be ablated successfully in the PA. In this report, we discuss 2 cases with idiopathic VTs originating from the PA.

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Case Report

Case 1

A 38-year-old woman with no previous cardiac history visited the hospital because of palpitations associated with dizziness. Physical examination revealed no abnormalities. The 12-lead surface ECG revealed normal sinus rhythm with frequent PVCs. The PVCs had an LBBB morphology, inferior-axis, QS pattern in lead I, and R/S ratio >1 in lead V4 (Figure 1(A)). The R-wave amplitude of the PVCs in the inferior leads were 1.9 mV(II), 1.9 mV(III), and 2.1 mV(aVF). Twenty-four-hour Holter monitoring revealed a repetitive nonsustained ventricular tachycardia (NSVT) of up to 12 beats with the same morphology as the PVCs. We suspected an idiopathic VT originating from the anterior-septal side of the RVOT. The patient was then referred to our institution for an electrophysiologic study and RFCA. Her laboratory data were normal, and the presence of structural heart disease was negated by echocardiography. Coronary angiog-

raphy did not reveal any significant stenosis of the left or right coronary arteries.

Electrophysiologic study and ablation procedure

After obtaining informed consent the procedure was performed with the patient under local anesthesia. Three catheters were inserted via the right femoral vein into the right atrium, His-bundle region, and RVOT under fluoroscopic guidance. One catheter was inserted via the internal carotid vein into the coronary sinus (CS). During the study, the presence of frequent spontaneous clinical VTs or PVCs was observed, so we did not use programmed stimuli to induce clinical VT or PVCs. While the mechanisms of those VTs and PVCs were not ascertained, we thought that triggered activity was the possible mechanism for those VTs and PVCs as is the usual case with idiopathic VTs with an LBBB morphology. At first we performed activation and pace mapping of the RVOT during the spontaneous clinical VT or PVCs. An endocardial activation preceding the QRS complex of the PVC by 30 msec

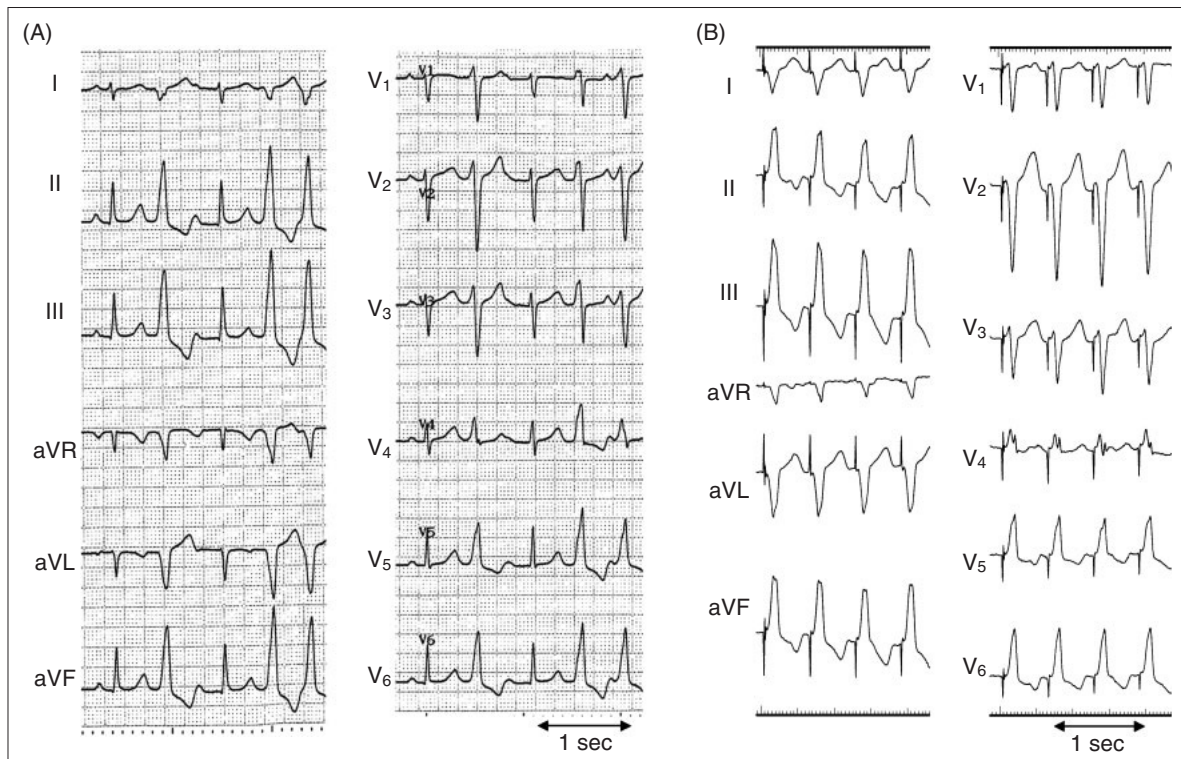


Figure 1

(A) A surface 12-lead ECG during sinus rhythm with spontaneous premature ventricular contractions (PVCs). The QRS morphology during the PVCs exhibited an inferior axis, left bundle branch block, QS pattern in lead I, and R/S ratio >1 in lead V4. The R-wave amplitude of the PVCs in leads II, III, and aVF was 1.9 mV, 1.9 mV, and 2.1 mV, respectively.

(B) A surface 12-lead ECG showing pace mapping from the anterior-septal side of the right ventricular outflow tract (RVOT) just under the pulmonary valve. The R-wave amplitude in leads V2 and V3 during pace mapping were lower than those of the clinical VT or PVCs.

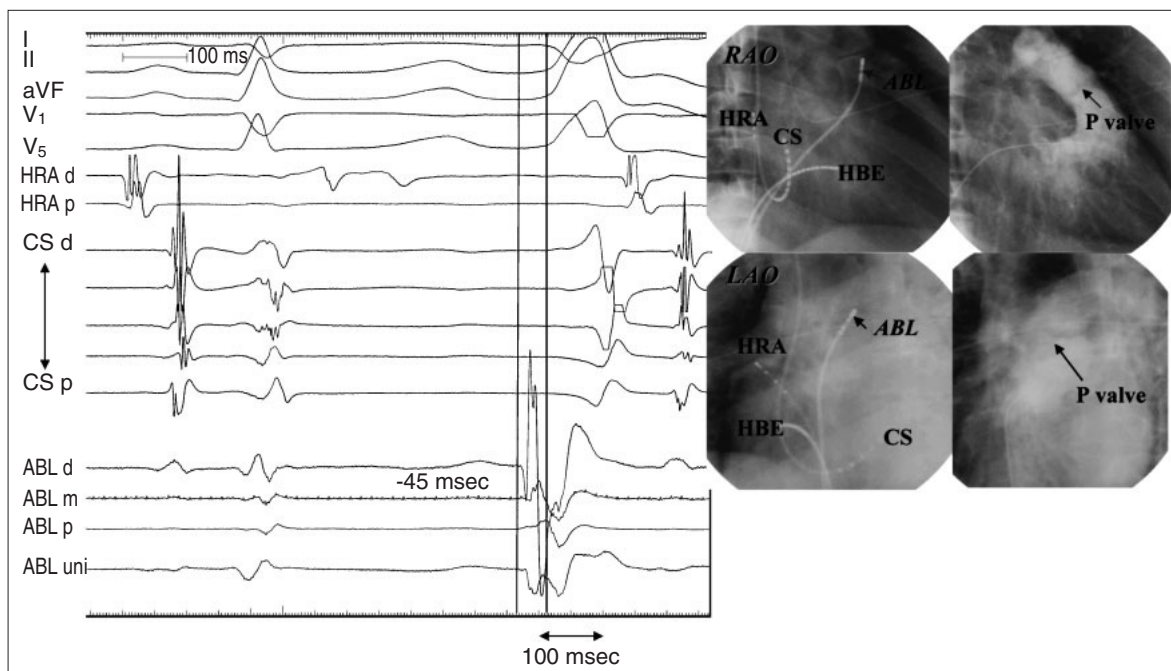


Figure 2

Left panel: A surface ECG and endocardial recordings during sinus rhythm and a clinical PVC. An endocardial activation preceding the QRS complex of the PVC by 45 msec was observed on the anterior-septal side of the PA about 1 cm above the pulmonary valve. The amplitude of the ventricular activation at the RFCA site in the PA during sinus rhythm was 0.04 mV. The amplitude of the ventricular activation at RFCA site during the PVC was 0.36 mV. Middle panel: Right and left anterior oblique fluoroscopic images of the catheter positions corresponding to the successful ablation site. Right panel: Fluoroscopic images of the right ventricular angiogram. RAO = right anterior oblique, LAO = left anterior oblique, HRA = high right atrium, HBE = his bundle electrode, CS = coronary sinus, ABL = ablation catheter, P valve = pulmonary valve

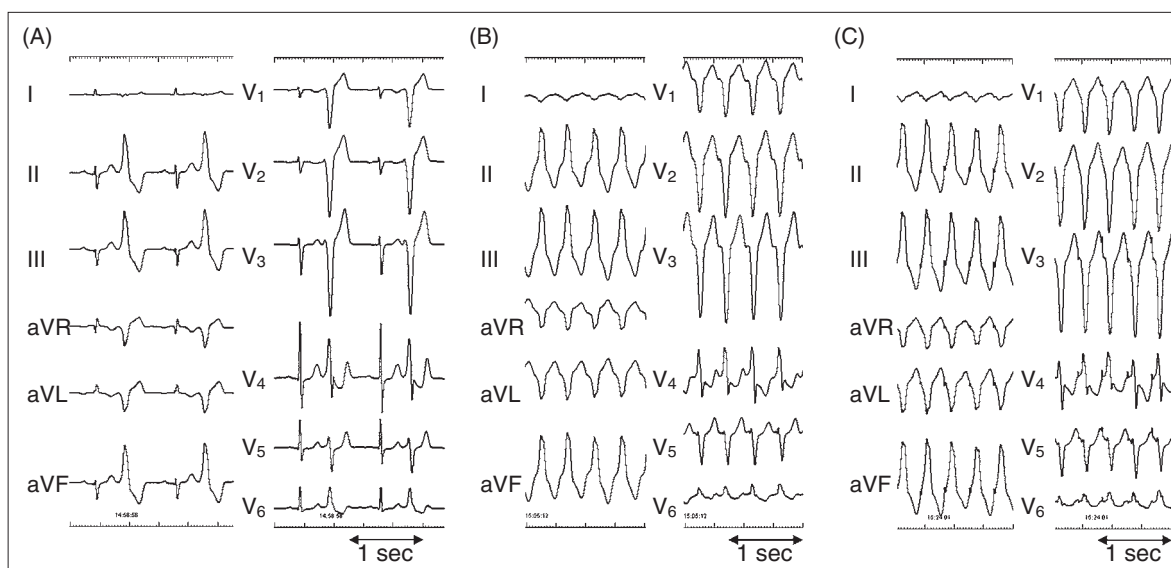


Figure 3

(A) A surface 12 lead ECG showing sinus rhythm with frequent PVCs. The morphology during the PVCs exhibited an inferior axis, left bundle branch block, QS pattern in lead I and R/S ratio >1 in lead V4. The R-wave amplitude of the PVCs in leads II, III, and aVF was 1.8 mV, 1.7 mV, and 1.8 mV, respectively.

(B) A surface 12 lead ECG during the clinical VT. The tachycardia cycle length was 330 msec. The morphology of the VT was the same as the PVCs.

(C) A surface 12 lead ECG showing pace mapping from the anterior wall of the PA just above the pulmonary valve. A perfect pace mapping score of 12/12 was obtained at that site.

was observed on the anterior-septal side of the RVOT just under the pulmonary valve. Bipolar pacing at this site revealed a high pace mapping score of 10/12 (**Figure 1(B)**). But two radiofrequency (RF) applications could not eliminate the VT or PVCs at this site. The R-wave amplitude in leads V2 and V3 during pace mapping was lower than that of the clinical VT or PVCs. Thus, we suspected a VT originating from the PA just above the pulmonary valve. We advanced the ablation catheter about 1 cm above the pulmonary valve and found a point on the anterior-septal side of the PA where the endocardial activation preceded the QRS complex of the PVC by 45 msec (**Figure 2**). At this site, bipolar pacing stimuli delivered from a conventional pacing unit using the maximal output (9.9 V/2.0 msec) could not capture the myocardium. A current delivery with a target temperature of 50 °C and a maximum power of 45 W using a conventional 4-mm-tip ablation catheter eliminated the PVCs after 10 seconds. Thereafter, the VT and PVCs were no longer inducible under an isoproterenol infusion.

During 13 months of follow-up, the patient has remained free from any VT recurrence.

Case 2

A 70-year-old woman had had palpitations due to PVCs and NSVT for 3 years even though she was taking 60 mg/day of metoprolol. The palpitations occurred during exercise or under conditions of stress. Twelve-lead surface ECG revealed normal sinus rhythm with frequent PVCs with an LBBB morphology, inferior-axis, QS pattern in lead I, and R/S ratio >1 in lead V4 (**Figure 3(A)**). The R-wave amplitude of the PVCs in the inferior leads were 1.8 mV(II), 1.7 mV(III), and 1.8 mV(aVF). Twenty-four-hour Holter monitoring revealed repetitive episodes of NSVT of up to 47 beats with the same morphology as the PVCs. The patient was then referred to our institution for an electrophysiologic evaluation and RFCA.

Physical examination revealed no abnormalities. The presence of structural heart disease was negated through a chest X-ray, echocardiography, and car-

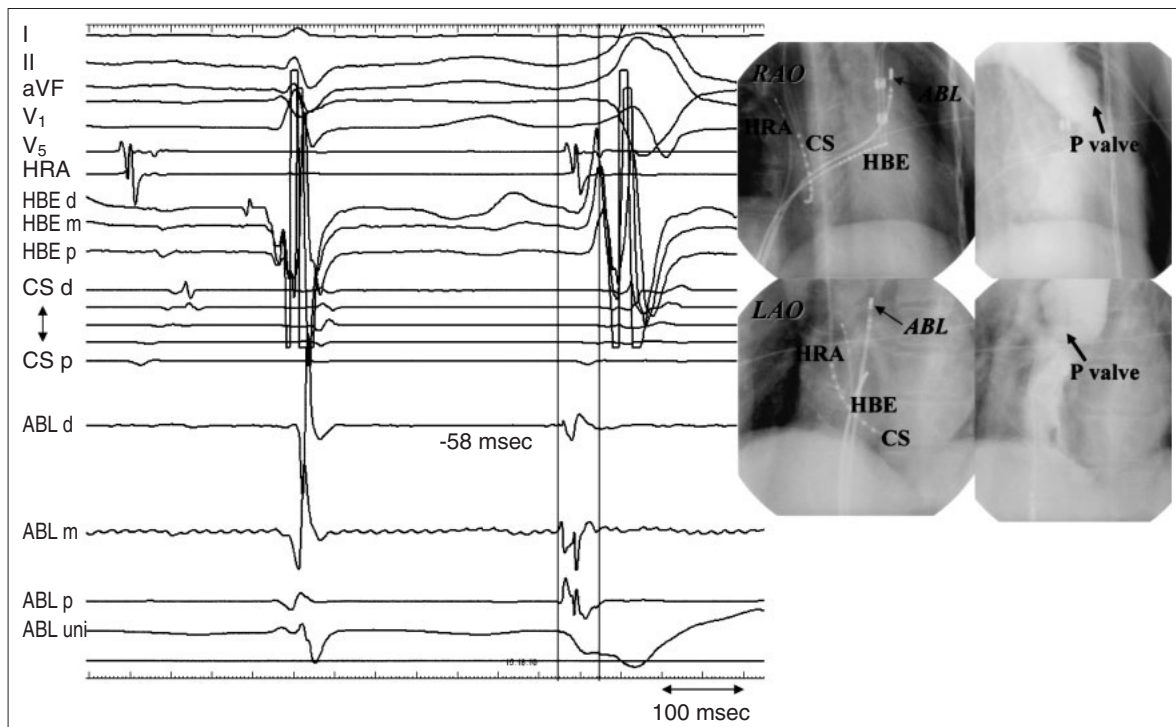


Figure 4

Left panel: A surface ECG and endocardial recordings during sinus rhythm and clinical PVC. An endocardial activation preceding the QRS complex of the PVC by 58 msec was observed at the anterior wall of the PA just above the pulmonary valve. The amplitude of the ventricular activation at that site during sinus rhythm was 3.47 mV. The amplitude of the ventricular activation at that site during the PVC was 0.26 mV. Middle panel: Right and left anterior oblique fluoroscopic images of the catheter position corresponding to the successful ablation site. Right panel: Fluoroscopic images of the right ventricular angiography. RAO = right anterior oblique, LAO = left anterior oblique, HRA = high right atrium, HBE = His bundle electrode, CS = coronary sinus, ABL = ablation catheter, P valve = pulmonary valve

diac catheterization including coronary and right and left ventricular angiography. Laboratory data were normal. Idiopathic VT or PVCs originating from the anterior-septal side of the RVOT were suspected.

Electrophysiologic study and ablation procedure

After obtaining informed consent the procedure was performed. The positions of catheters were the same as in Case 1. An electroanatomical mapping system (CARTO, Biosense Webster) was used to conduct activation and voltage mapping. During the study, the presence of frequent spontaneous clinical VTs or PVCs were observed. Activation mapping was performed from the RVOT and PA just above the pulmonary valve during the spontaneous clinical VT (**Figure 3(B)**) or PVCs. An endocardial activation preceding the QRS complex of the PVCs by 58 msec was observed on the anterior wall of the PA just above the pulmonary valve (**Figure 4**), and a perfect pace mapping score of 12/12 was obtained during the stimulation at this site (**Figure 3(C)**). The CARTO voltage mapping revealed that the earliest ventricular activation was recorded in a low voltage area (**Figure 5**). It suggested that the earliest ventricular activation site was in the PA. During the mapping at the site, a bump phenomenon occurred. Therefore,

we thought the site was the focus of the PVCs or VT. Three RF applications with a target temperature of 50 °C and maximum power of 45 W using a conventional 4-mm ablation catheter for 90 seconds at the site resulted in the noninducibility of the PVCs or VT even after the infusion of isoproterenol. During 6 months of follow-up, the patient has remained free from any VT or PVC recurrence.

Discussion

Most idiopathic VTs with an LBBB morphology have their origin on the septal side of the RVOT and some originate from the free wall of the RVOT, LVOT, or ASC.¹⁻¹⁰ However, there are some cases in which we cannot eliminate those arrhythmias from any of those sites. In recent years, idiopathic VTs originating from the PA have been described.¹⁻³ Timmermans et al. reported that the origin of idiopathic VTs with an LBBB morphology can be from the root of the PA and explained that the site of the VT origin within the PA may be in the myocardial tissue in or around the PA. They referred to embryology as a possible explanation. The embryonic mammalian OT is surrounded by myocardium. The proximal outflow tract myocardium

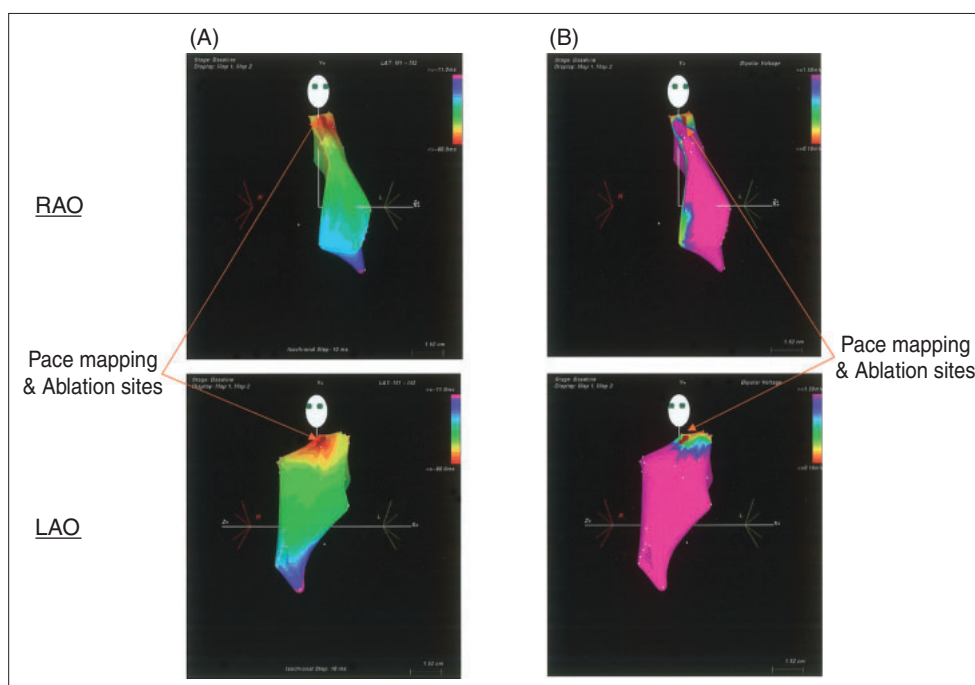


Figure 5

Electroanatomic mapping of the RVOT and PA just above the pulmonary valve. The activation mapping (right panel) revealed that the earliest ventricular activation was observed at the anterior wall of the PA just above the pulmonary valve. Voltage mapping (left panel) revealed that the earliest ventricular activation was recorded in the low voltage area.

becomes ventricularized by incorporation into the right ventricle, whereas the myocardium of the distal outflow tract does not disappear completely and remnants persist that may provide the substrate for idiopathic VTs.¹⁾ In both cases in our report, sharp or dull potentials were recorded at the successful RFCA sites in the PA. Those potentials may indicate the presence of muscular tissue in or around the PA. In Case 2, CARTO voltage mapping showed that the successful RFCA site was located in a low voltage area in the PA. This result would suggest that even though the amount of muscular tissue in the PA was low, that muscular tissue could have provided the substrate for the idiopathic VTs. Timmermans et al. also reported that the successful RFCA sites for 6 idiopathic VTs originating from the PA were several centimeters above the pulmonary valve.¹⁾ Sekiguchi et al. reported that the successful RFCA sites for 24 idiopathic VTs originating from the PA were 1.18 ± 0.43 cm above the pulmonary valve.²⁾ Their report suggested that ectopic muscular tissue can exist a few centimeters above the pulmonary valve. The results of their study corresponded to our findings in Case 1 of this report.

How do we predict the origin of idiopathic VTs with an LBBB morphology? In both of our cases, we suspected that the origin of the VTs was located on the anterior-septal side of the RVOT. Even though there are some ECG algorithms for identifying the optimal ablation site for idiopathic VTs,¹⁻⁹⁾ it is difficult to distinguish between those VTs originating from the PA and those from the RVOT or ASC. Timmermans et al. reported that in 6 idiopathic VTs originating from the PA, 3 VTs had an R/S wave amplitude ratio > 1 in lead V4, 2 VTs in V3, and 1 VT in V6.¹⁾ Sekiguchi et al. reported that the R-wave amplitude in the inferior leads and R/S wave amplitude ratio in V2 of idiopathic VTs originating from the PA were significantly larger than those in the RVOT.²⁾ Ouyang, et al. reported that the R/S wave amplitude ratio in V1 or V2 was larger in idiopathic VTs originating from the RVOT than in those from the ASC.⁴⁾ In both cases in our report, the R-wave amplitude in the inferior leads was comparatively large, and the R/S wave amplitude ratio in V1 and V2 was low. A large R-wave amplitude in the inferior leads may indicate that the PA or ASC is the origin of the idiopathic VT with an LBBB morphology. However, there were variations in the R/S wave amplitude ratio in the precordial leads, which we also found in our two cases, and thus it was difficult to make a distinction between the idiopathic VTs originating from the PA and those from the ASC by using surface ECG.

Pace mapping is also useful for determining the RFCA site. In Case 2, the VTs could be eliminated at the anterior wall of the PA just above the pulmonary valve where a perfect pace mapping score of 12/12 was obtained. However, in Case 1, the VTs and PVCs could not be eliminated at the anterior-septal side of the RVOT just under the pulmonary valve even though a high pace mapping score of 10/12 was obtained. Finally, we succeeded in eliminating the VTs on the anterior-septal side of the PA 1 cm above the pulmonary valve. Our case report may suggest that if a perfect pace mapping score is not obtained in the RVOT just under the pulmonary valve, the PA should be mapped. If conventional pacing can not capture the PA myocardium, Sekiguchi et al. reported that high-output pacing with a transesophageal pacing unit is useful.²⁾ Regrettably, we did not have a high-output pacing system, so we had to abandon the use of pace mapping in Case 1.

Our case report suggested that in idiopathic VTs with an LBBB morphology, the RVOT should be mapped first and if no early activation time during the VT or optimal pace mapping sites are found in the RVOT, detailed mapping in the PA should be performed, especially for VTs with larger R-wave amplitudes in the inferior leads. If a ventricular potential is recorded in the PA, the potential may indicate there is ectopic muscular tissue which may provide the substrate for idiopathic VTs.

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